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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/579,894	05/26/2000	Kalle Saksela	0933-0159P	9956

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EXAMINER
CELSA, BENNETT M

ART UNIT	PAPER NUMBER
1639	18

DATE MAILED: 01/16/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

file copy

Office Action Summary

Application No.	Applicant(s)
09/579,894	Saskela et al.
Examiner	**Art Unit**
Bennett Celsa	1639

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *three* MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136 (a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1) ☒ Responsive to communication(s) filed on *Nov 16, 2002* .

2a) ☐ This action is **FINAL.** 2b) ☒ This action is non-final.

3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11; 453 O.G. 213.

Disposition of Claims

4) ☒ Claim(s) *1-19* is/are pending in the application.

4a) Of the above, claim(s) *5-16* is/are withdrawn from consideration.

5) ☐ Claim(s) _____ is/are allowed.

6) ☒ Claim(s) *1-4 and 17-19* is/are rejected.

7) ☐ Claim(s) _____ is/are objected to.

8) ☐ Claims _____ are subject to restriction and/or election requirement.

Application Papers

9) ☐ The specification is objected to by the Examiner.

10) ☐ The drawing(s) filed on _____ is/are a) ☐ accepted or b) ☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11) ☐ The proposed drawing correction filed on _____ is: a) ☐ approved b) ☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12) ☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13) ☐ Acknowledgement is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a) ☐ All b) ☐ Some* c) ☐ None of:

1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. _____ .
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

*See the attached detailed Office action for a list of the certified copies not received.

14) ☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. § 119(e).

a) ☐ The translation of the foreign language provisional application has been received.

15) ☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s). _____
4) ☐ Interview Summary (PTO-413) Paper No(s). _____
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other:

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 11/6/02 has been entered.

NOTE: for future correspondences, the location of the present application is ART UNIT 1639.

2. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Status of the Claims

Claims 1-19 are currently pending.

Claims 1-4 and 17-19 are under consideration.

Claims 5-16 are withdrawn from consideration as being directed to nonelected subject matter. A complete reply to the final rejection must include cancellation of nonelected claims or other appropriate action (37 CFR 1.144) See MPEP § 821.01.

Withdrawn Objection (s) and/or Rejection (s)

Applicant's amendment has overcome the indefinite rejection of claims 1-4 and 17-19 in the prior office action.

Applicant's amendment has overcome the new matter rejection of claims 17-19 in the prior office action.

New Objection (s) and/or Rejection (s)

3. Claims 17-19 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

A. In claim 17 (and claims dependent thereon), "the HIV-1 Nef protein" lacks clear antecedent basis.

B. In claim 17 (and claims dependent thereon), the phrase "targeted to the HIV-1 new protein" is indefinite since there is no antecedent basis for "targeted" in claim 1. Amending claim 1 to recite "desired ligand bind properties *to a target*" will overcome this rejection.

Outstanding Objection(s) and/or Rejection (s)

4. Claims 1-4 are rejected under 35 U.S.C. 102(b) as anticipated by or, in the alternative, under 35 U.S.C. 103(a) as obvious over Lee et al. Embo J. Vol. 14, No. 20 pages 5006-5015 (1995)..

Lee et al. discloses a method of producing SH3 domains from the RT-loop region of different SH3 domains (e.g. from different SH3-kinases) . Lee produces said SH3 domains by first mutating some residues of the RT-loop of the different SH3 domains, e.g. page 5010, Fig. 4. The "collection" of mutant RT-loop region is obtained from a library of cDNA (e.g. a collection of 2 or more) . "DNA fragments encoding" SH3 domains containing a "randomized RT-loop

(RRT-SH3 domains)" are taught by the reference e.g. by use of cDNA encoding the kinase (e.g .human Hck) and polymerase chain reaction (e.g. amplification using primers) with "cloning" utilizing a "plasmid *vector*" to generate the recombinant library (e.g. see Lee et al. page 5013, right column). The RT-loop mutated region is then affinity purified to identify the mutant RT-loop peptide that binds to the PXXP motif of e.g., Nef with specificity and affinity; as well as the binding of the other "artificial" (e.g. unnaturally occurring) SH3 domains to their "desired ligands". In this regard the reference discloses "randomized" substitutions (one or a *combination e.g. 2, 3*) of amino acid substitutions within the RT loop, and specifically within non-conserved (e.g. "variable" regions), and *preferably* including one or more substitutions (e.g. within a specific kinase or among a library of kinases) within a span which "*comprise* six amino acids that immediately follow a conserved stretch of amino acids having an ALYDY consensus sequence". (See e.g. Fig. 4 teaching both conserved and non-conserved amino acids of the RT-loop of kinases and Table I teaching the construction of a library (e.g a collection) of different kinases having "artificial SH3 domains having desired ligand binding properties" "comprising randomized RT-loops" wherein the collection of SH3 domains contain one or more "random" amino acid substitutions that comprise a hexapeptide sequence "that immediately follows a conserved stretch of amino acids having an ALYDY" (e.g. ...**(AL) YDY** hexapeptide DLS ...). With respect to SH3 binding and specificity (e.g. w/r to differential binding of SH3 containing kinases e.g. Hck and Fyn) to HIV-I, the Lee reference teaches that **"distinct specificity lies in a variable loop, the 'RT loop', positioned close to conserved SH3 residues implicated in the binding of**

proline-rich (PXXP) motifs" (emphasis provided) . See ABSTRACT. It is considered that the different mutations of the different SH3 regions of the different kinases is the same to the claimed randomized RT-loop domains or would have been obvious to make into a random collections in view of the Lee's disclosure as to the different amino acids that can be mutated in the different SH3 domains of the SH3 wild type, particularly within the non-conserved regions of the RT-loop motif.

Discussion

Applicant's amendment and arguments relating to the above 102/103 rejection was considered but deemed nonpersuasive for the following reasons. Initially, it is noted that the above 102/103 rejection was modified in order to address the newly amended claim limitations.

Applicant argues that "[I]n Lee et al. the RT-loop of Fyn-SH3 was modified to resemble the Hck-SH3. Thus, Lee et al. do not disclose the generation of randomized new sequences for RT-loop domains, rather they simply replace the RT-loop of Fyn-SH3 with another naturally occurring RT-loop sequence, that of Hck-SH3."

This argument is not found persuasive for several reasons.

First, the Lee et al. reference is not limited to one mutant SH3 domain (e.g. the Fyn SH3 mutant referred to by applicant) but extends to the making of multiple SH3 kinase domains containing multiple mutant RT-loop w/n non-conserved regions. Applicant has thus failed to appreciate the Lee reference teaching as a whole.

Secondly, the making of a mutant SH3 which *differs from the wild SH3 region* of a kinase would constitute a "new sequence" for one or more RT-loop domains as taught by the reference.

Thirdly, as pointed out in the 102/103 rejection above, the Lee reference clearly teaches (e.g. through example) and suggests (e.g. through explicit statements: e.g. see abstract) the making of artificial (e.g. differing from the wild type) SH3 domains of different kinase that contain random (e.g one or more amino acid substitutions) within the non-conserved regions (particularly a hexapeptide region) of the RT-loop region.

Applicant argues (citing specification discussion on page 5, lines 6-21) that "the present inventors have found that by using the presently recited method of random generation of the RT-loop sequence combined with affinity selection, instead of merely mimicking known SH3 domains, one can generate SH3 domains with specifically desired binding properties, such as unnaturally high affinity for specific proteins. Applicant further argues that there is no disclosure or suggestion in Lee et al. of a means of generating any but naturally occurring SH3 binding domains or of a method of generating artificial SH3 domains having desired binding properties.

Applicant's argument is not persuasive since it fails to appreciate both the specific teaching of the Lee et al. reference (through its examples) and the Lee reference teaching taken as a whole. As recited in the 102/103 rejection, the Lee et al. reference provides means for making SH3 domains (e.g. recombinant libraries employing cDNA, PCR, mutagenesis and recombinant libraries using plasmid cloning) and screening for desired (e.g. ligand- binding) clones; which SH3 domains are "artificial" by differing from the wild type due by one or more

amino acid substitutions in the non-conserved portion (e.g. variable region) of the RT loop for one or more SH3 kinases.

Dr. Saksela's statements in the declaration regarding the Lee et al. reference teaching were considered but deemed nonpersuasive for the following reasons. In Dr. Saksela's opinion, the Lee reference failed to "hint that an artificial SH3 domain able to bind to HIV Nef could be made by any other way than mimicking the RT-loop sequence of the Hck tyrosine kinase". In contrast Dr. Saksela states that "the present invention *discloses* (emphasis provide) that specific binding to HIV Nef can be achieved by combinations of six RT-loop amino acids that bear no similarity whatsoever with the corresponding sequence in Hck or in any other natural SH3 domain".

However, to the extent that Dr. Saksela's arguments address limitations not present in the claim (binding to HIV Nef; combinations of six RT-loop amino acids etc.) this argument is simply not persuasive. Additionally, Dr. Saksela focuses on the specific Lee example, which fails to consider other portions of the Lee et al. document pointed to in the rejection e.g. Dr. Saksela fails to consider the reference teaching as a whole to one of ordinary skill in the art. Further, Dr. Saksela's statement regarding the Lee et al. examples strategy of "mimicking nature" fails to address the fact that the reference teaches the making of "artificial" SH3 domains (e.g. artificial sequences which do not naturally occur) by "randomization" (e.g. the creation of artificial peptides having one or more amino acid substitutions). .

Applicant's Preliminary Response was considered but deemed nonpersuasive for the following reasons.

Applicant's preliminary response asserts that the terms "artificial"; "randomization" and "library" are not indefinite as allegedly asserted by the Examiner (e.g. in the Advisory action). Applicant further states that these terms are "well-known in the art and used with the same well-known meanings in the context of the present invention".

Initially, it noted that applicant has misconstrued what was stated by the Examiner in the Advisory Action. The examiner did not state that the above terms were indefinite; but merely meant that the claimed terminology (since not specifically defined in the specification) will be afforded its *broadest art-accepted definition.* Accordingly, the term "artificial SH3 domains" would be construed as domains that are not present in nature (e.g. differ from the wild type). The term "randomized RT loops" would represent a wild type RT loop containing "one or more amino acid substitutions" thus resulting in an "artificial" SH3 domain e.g. an SH3 domain not present in nature due to its non-wild type loop. Finally, the term "library" in a broadest art-accepted context represents a collection containing a plurality (e.g. 2 or more) of members; which in the case of a "recombinant library" is produced recombinantly. The Examiner's interpretation is believed to be consistent with the prior art, statements made in Dr. Saksela's Declaration; and references provided in the Preliminary Response.

Applicant further argues that Lee et al. teaches "designed and specifically directed mutagenesis" . This is not persuasive since the Lee et al. Reference method generates libraries

(e.g. two or more) by recombinant means and thus is within the scope of applicant's claimed invention. Applicant's argument that a "library" is made of a *great number* of recombinant vectors is not supported by the specification or the prior art and is not consistent with the broadest interpretation (consistent with the prior art) of this term.

Applicant further argues that Lee et all does not produce randomized libraries e.g. by use of site directed mutagenesis.

Applicant's argument is not commensurate to the claimed invention with is drawn to artificial SH3 domains comprising "randomized RT-loop(s)" (e.g. "creation of new peptide having one or more amino acid substitutions: see Declaration at page 5) by "generating recombinant libraries" (e.g. collection of two or more members by recombinant means) which is taught by the Lee reference.

Accordingly, the above revised 102/103 rejection, as modified, is hereby maintained.

5. Claims 1-4 and 17-19 are rejected under 35 U.S.C. 103(a) as being unpatentable over Lee et al. Embo J. Vol. 14, No. 20 pages 5006-5015 (1995) and Sparks et al. J. Biol. Chem. Vol. 269, No. 39 (9/1994) pages 23853-23856.

Lee et al. discloses a method of producing SH3 domains from the RT-loop region of different SH3 domains (e.g. from different SH3-kinases) . Lee produces said SH3 domains by first mutating some residues of the RT-loop of the different SH3 domains, e.g. page 5010, Fig. 4. The "collection" of mutant RT-loop region is obtained from a library of cDNA (e.g. a collection

of 2 or more) . "DNA fragments encoding" SH3 domains containing a "randomized RT-loop (RRT-SH3 domains)" are taught by the reference e.g. by use of cDNA encoding the kinase (e.g .human Hck) and polymerase chain reaction (e.g. amplification using primers) with "cloning" utilizing a "plasmid *vector*" to generate the recombinant library (e.g. see Lee et al. page 5013, right column). The RT-loop mutated region is then affinity purified to identify the mutant RT-loop peptide that binds to the PXXP motif of e.g., Nef with specificity and affinity; as well as the binding of the other "artificial" (e.g. unnaturally occurring) SH3 domains to their "desired ligands". In this regard the reference discloses "randomized" substitutions (one or a *combination e.g. 2, 3*) of amino acid substitutions within the RT loop, and specifically within non-conserved (e.g. "variable" regions), and *preferably* including one or more substitutions (e.g. within a specific kinase or among a library of kinases) within a span which "*comprise* six amino acids that immediately follow a conserved stretch of amino acids having an ALYDY consensus sequence". (See e.g. Fig. 4 teaching both conserved and non-conserved amino acids of the RT-loop of kinases and Table I teaching the construction of a library (e.g a collection) of different kinases having "artificial SH3 domains having desired ligand binding properties" "comprising randomized RT-loops" wherein the collection of SH3 domains contain one or more "random" amino acid substitutions that comprise a hexapeptide sequence "that immediately follows a conserved stretch of amino acids having an ALYDY" (e.g. ...**(AL) YDY** hexapeptide DLS ...). With respect to SH3 binding and specificity (e.g. w/r to differential binding of SH3 containing kinases e.g. Hck and Fyn) to HIV-I, the Lee reference teaches that **"distinct specificity lies in a variable loop,**

the 'RT loop', positioned close to conserved SH3 residues implicated in the binding of proline-rich (PXXP) motifs" (emphasis provided) . See ABSTRACT. It is considered that the different mutations of the different SH3 regions of the different kinases is the same to the claimed randomized RT-loop domains or would have been obvious to make into a random collections in view of the Lee's disclosure as to the different amino acids that can be mutated in the different SH3 domains of the SH3 wild type, particularly within the non-conserved regions of the RT-loop motif.

The Lee et al. reference teaching differs from the presently claimed invention (e.g. new claims 17-19) since it fails to explicitly teach generating "artificial Hck-SH3" libraries by randomizing (e.g. with all 20 natural amino acids) the non-conserved hexapeptide 69-74 (EAIHHE) RT-loop sequence of Hck (or related SH3 kinases) to obtain completely random libraries comprising 20^6 (e.g. 20x20x20x20x20x20) artificial Hck-SH3 proteins differing from the wild type at hexapeptide 69-74 (EAIHHE) for subsequent ligand screening (e.g. with HIV-I Nef) and selection of artificial Hck-SH3 proteins containing "optimum" motifs.

However, the Lee et al. reference further teaches that HIV-I Nef protein binds to the SH3 domains of a subset of Src family kinases (including Hck and Fyn); and the SH3 binding capacity of Nef is necessary for optimal spread of HIV-I infection (e.g. via replication). Accordingly, blocking the interaction (e.g. via use of competitive inhibitors) between the HIV-Nef protein and the Src family kinases (e.g. Hck and Fyn) may be therapeutic for HIV infection. See e.g. page 5006 right column to page 5007. The Lee et al .reference further teaches that, w/r to specificity

and binding of HIV-I Nef protein to the SH3 domain of Src family kinases, "distinct specificity lies in a variable loop, the 'RT loop', positioned close to conserved SH3 residues implicated in the binding of proline-rich (PXXP) motifs" e.g. at hexapeptide 69-74 (EAIHHE) non-conserved peptide region of Hck (and the corresponding position w/r to the other Src family kinases); and thus the development of artificial SH3 protein analogs which preferentially bind the HIV-I Nef protein may be therapeutic in preventing HIV infection. E. g. See abstract; and page 5013.

Accordingly, the Lee et al. reference provides motivation to one of ordinary skill in the art to make recombinant libraries (using the Lee reference method) that comprise randomization of the non-conserved (e.g. variable) RT loop hexapeptide 69-74 (EAIHHE) peptide sequence of Hck or the corresponding region in other Src family kinases in order to screen such libraries for potential competitive inhibitors useful in treating HIV infection. One would be motivated to completely randomize the hexapeptide variable RT loop region in order to obtain the largest possible library (e.g. a completely random library comprising 20^6 (e.g. 20x20x20x20x20x20) artificial Hck-SH3 proteins or other artificial Src family kinase proteins) for screening and thus *optimizing* the likelihood of finding therapeutically useful competitive inhibitors.

Thus, it would have been obvious to one of ordinary skill in the art at the time of applicant's invention, in light of the Lee reference teaching alone, to generate "artificial Hck-SH3" peptide (or other Src family kinase peptide) libraries by randomizing (e.g. with all 20 natural amino acids) a hexapeptide 69-74 (EAIHHE) of Hck (or the corresponding region of a related Src family protease) to obtain a complete random library comprising 20^6 artificial Hck-

SH3 proteins differing from the wild type at hexapeptide 69-74 (EAIHHE) since the Lee reference suggests the making of competitive inhibitors of HIV infection by modifying the amino acids in the variable hexapeptide (69-74) region of Hck (or other Src family kinases) to generate inhibitors. Additionally, the making of the largest library (e.g by complete randomization of each amino acid) for screening potential HIV-I inhibitors represents mere optimization.

Additionally, the Sparks et al. reference teaches the utilization of "biased peptide libraries" or , preferentially "random peptide libraries" (e.g. all 20 amino acids), including 7mer/8mer peptide libraries, via phage display, as a means for making and screening Src SH3 ligands for developing "antagonists of Src SH3 interactions with SH3-binding proteins" . See abstract; and entire article.

Accordingly, the Sparks et al. reference provides further motivation to make and screen "random peptide libraries" for developing "antagonists of Src SH3 interactions with SH3-binding proteins" which can be useful to treat HIV infection.

Thus, it would have been obvious to one of ordinary skill in the art, in view of the combined teaching of the Lee and Sparks references, to generate "artificial Hck-SH3" (or other Src family kinase proteins) libraries by making "random peptide libraries" comprising the hexapeptide 69-74 (EAIHHE) peptide sequence (or corresponding sequence) to obtain a complete random library, using either the Lee (recombinant) or Sparks (phage display) method of library generation in order to screen for potential HIV therapeutics.

Discussion

Applicant's arguments directed against the above obviousness rejection were considered but deemed nonpersuasive for the following reasons.

Initially, it is noted that arguments already addressed by the Examiner above will not be reiterated here.

Applicant argues that the Sparks et al. Reference addresses the PXXP motif (e.g. of the ligand) which differs from the presently claimed emphasis on the SH3 domain (e.g. of the enzyme receptor).

In response to applicant's arguments against the references (e.g. Sparks et al.) individually, one cannot show nonobviousness by attacking references individually where the rejections are based on combinations of references. See *In re Keller*, 642 F.2d 413, 208 USPQ 871 (CCPA 1981); *In re Merck & Co.*, 800 F.2d 1091, 231 USPQ 375 (Fed. Cir. 1986).

Applicant further argues that the Lee et al. and the Sparks et al. reference teachings "contradict each other" since the Lee et al. reference concentrates on varying the variable RT-loop portion of and SH3 of a Src family kinase (e.g. Hck-SH3) while the Sparks et al. reference concentrates on varying amino acids flanking the PXXP motif (e.g. of the enzyme ligand). Applicant further argues that the Lee et al. And Sparks references target different "important regions for binding" (SH3 RT-loop of the enzyme receptor vs. the PXXP motif of the ligand) which do not interact with each other; thus making these references non-combinable to one of ordinary skill in the art.

Applicant's analysis is not convincing since it is misguided in the following respects.

First, applicant is failing to consider the reference teachings as a whole to one of ordinary skill in the art. Additionally, applicant is not addressing the substantive aspects of the above rejection regarding the teachings of the references and why one would be motivated to combine such reference teachings. In response to applicant's argument that there is no suggestion to combine the references, the examiner recognizes that obviousness can only be established by combining or modifying the teachings of the prior art to produce the claimed invention where there is some teaching, suggestion, or motivation to do so found either in the references themselves or in the knowledge generally available to one of ordinary skill in the art. See *In re Fine*, 837 F.2d 1071, 5 USPQ2d 1596 (Fed. Cir. 1988)and *In re Jones*, 958 F.2d 347, 21 USPQ2d 1941 (Fed. Cir. 1992). In this case, as discussed in the rejection above, motivation to combine is provided by both the reference teachings and in the knowledge generally available to one of ordinary skill in the art. For example, as pointed out in the rejection above, "the Lee et al. reference provides motivation to one of ordinary skill in the art to make recombinant libraries (using the Lee reference method) that comprise randomization of the non-conserved (e.g. variable) RT loop hexapeptide 69-74 (EAIHHE) peptide sequence of Hck or the corresponding region in other Src family kinases in order to screen such libraries for potential competitive inhibitors useful in treating HIV infection. One would be motivated to completely randomize the hexapeptide variable RT loop region in order to obtain the largest possible library (e.g. a completely random library comprising 20^6 (e.g. 20x20x20x20x20x20) artificial Hck-SH3 proteins

or other artificial Src family kinase proteins) for screening and thus *optimizing* the likelihood of finding therapeutically useful competitive inhibitors. "

Accordingly, the above rejection is hereby maintained.

General information regarding further correspondence

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Examiner Celsa whose telephone number is (703) 305-7556.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Andrew J. Wang (art unit 1639), can be reached at (703)306-3217.

Any inquiry of a general nature, or relating to the status of this application, should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Bennett Celsa (art unit 1639)

January 15, 2003

BENNETT CELSA
PRIMARY EXAMINER